ON THE

STATE OF THE CIRCULATION IN ACUTE DISEASES.

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(Communicated by the Editor.)

The attention of all observers who have made use of the sphygmograph has been hitherto directed rather to its clinical application as an aid to diagnosis and prognosis than to its employment for the solution of any physiological problems relating to the vascular system. This has been inevitable from the confusion which has prevailed among physiologists as to the explanation of the waves seen in the sphygmographic tracing, and the inherent difficulty of the mechanical theory of the pulse. Yet the sphygmograph would probably have its greatest value as applied to such general questions, rather than as an addition to the ordinary means of clinical research, since, however valuable its indications often are in reference to individual cases, its employment requires an expenditure of time which but few persons would be able to bestow.

In the 'Journal of Anatomy and Physiology' for November, 1873, I have discussed the cause of the secondary waves of the pulse, and in the present paper I shall assume the results there obtained, so far as concerns their practical application; for, although my view differs from that hitherto most generally received as to mechanical causes, yet as to the inferences to be drawn, with regard to the state of the vessels and the mode of

action of the heart, it is not inconsistent with it. There are two secondary waves whose interpretation is practically important—the first secondary or tidal wave, and the principal secondary or dicrotic wave. As to the tidal wave, I believe that its separation from the primary wave is due to acquired velocity in the sphygmograph, which causes the first pointed summit, or so-called "percussion-wave," to rise above the level of the true pulse-wave. I agree, however, with most writers in considering that the expansion of the tidal wave does actually denote a prolonged flow of blood, and therefore a protracted contraction of the heart, and that in many cases it nearly coincides with the corresponding part of the true pulse-wave. Experimentally its development is found to be increased, both by increase of tension and by diminution of elastic distensibility, both which conditions tend to make the heart's action more slow and laborious.

The relative magnitude of the tidal wave, as compared with the primary, is greatest in arteries near to the heart, such as the carotid, and becomes less on approaching the periphery. Thus, in a healthy pulse, in which the tidal wave in the radial artery is small, it may be absent in the arteries of the foot, but it remains very manifest there in the pulse of atheroma or of Bright's disease. In that case it follows the primary wave at about the same interval as in the radial, but in pulses in which it is small from the first, in receding from the heart it tends to become lost upon the preceding primary wave, a change which is just the opposite to that which would occur if the common theory were true, that it is a wave transmitted in the artery with a less velocity than the primary wave. This change of relative magnitude results from a change in the shape of the true pulse-wave, which is analogous to that in a wave of the sea as it reaches the shore; for in that case the wave velocity is greater the greater the depth of water, and thus the crest of the wave outstrips its base, until at length it curls over and breaks. Similarly in the pulse the wave velocity is greater the greater the tension, and therefore the crest of the pulse-wave moves rather more swiftly than its other portions. Thus, as the wave proceeds, its front retains its steepness, or grows even steeper, but the slope of its descending curve becomes more gradual, and thus the tidal wave, which then comes to correspond with this

descending curve, shows a gradually diminishing magnitude as compared with the primary.

The primary wave, when separated from the tidal, denotes the steepness of front of the true pulse-wave, and, therefore, by inference, the initial vigour of the heart's contraction. I do not, therefore, greatly object to the term "percussion-wave," which has been commonly applied to it, although I believe that no percussion takes place except in the communication of motion from the artery to the sphygmograph, and that what occurs even there is not percussion, in the strict sense of the word.

As to the dicrotic wave, I think that the common theory, which attributes it to a recoil produced by the closure of the aortic valves, is only partially true, and that two causes mainly contribute to its formation. The first of these is a recoil from the aortic valves, as the common theory supposes. I think, however, that, as to the mode of origin of this, sufficient regard has not generally been paid to the effect of the inertia of the fluid. It is evident that all changes of state in the aorta close to the valves will be propagated as waves to the periphery, although these waves may become modified as they proceed. Considering, then, the state of things in the aorta near to the valves at the moment that the ventricle ceases to contract, the blood will for an instant continue to flow away in consequence of its acquired velocity, and, the propelling force behind having ceased, the pressure will fall. As soon as the acquired velocity is checked by the pressure in front there is a slight retrograde motion, which closes the aortic valves, and, being reflected from them, causes a second forward wave of increased pressure and expansion.

The preceding retrograde motion and fall of pressure is also as a wave propagated forwards, although the motion of individual particles is at first backwards, and thus it does not involve any reflection from the periphery. As soon as it has proceeded a short distance from the heart it consists no longer of any actual retrograde movement of particles, but only of a diminution of forward velocity.

Since, therefore, the fall and subsequent elevation of pressure, which constitutes the first cause of the dicrotic wave, depends on acquired velocity in the fluid, dicrotism is increased if the density of the fluid be increased, or if the tension in the

tubes, by which such velocity is checked, be diminished. In the latter case there will be a longer interval between the cessation of the heart's contraction and the second rise of pressure. Thus, the commencement of the dicrotic wave corresponds to a point always an instant later than the end of the systole, but this instant is a shorter one in a pulse of high tension than in one of low. Even in the absence of the aortic valves it is conceivable that the retrograde movement of fluid, meeting with the onward flow from the auricle, might cause a second rise of pressure, to be again transmitted as a forward wave of expansion. In that case, however, it could hardly call out a descending series of similar waves, which may happen if the first reflection occurs from elastic valves.

Such is one cause of the dicrotic wave, and it will be seen that even this does not depend solely upon the aortic valves, but that it is an oscillation or forward and backward movement, due directly to the inertia of the fluid. But there are several reasons for thinking this cause insufficient in itself. 1. With an artificial heart and schema of bifurcating elastic tubes there may be obtained, in the entire absence of the aortic valves, not only a considerable dicrotic wave, but a series of succeeding waves, although all are smaller than if the valves were present. 2. These waves occur to some extent even without any cessation of the heart's contraction, and, therefore, without any retrograde movement of fluid at all. 3. If a tracing be taken from the schema close to the valves, the secondary waves are much less marked than they are at a distance. The same thing has been shown from experiments on animals in tracings representing the pressure in the aorta close to the heart, for the dicrotic wave, though visible there, is but little marked. So far as it depends on the cause already mentioned, it would, of course, be greatest near its origin at the valves.

In the paper before referred to, I have endeavoured to show that the effect of the inertia of the arterial walls would be to set up an oscillation of expansion and contraction in the largest arteries, which would contribute to the formation of the dicrotic wave. The mode in which I suppose this inertia to come into play may be illustrated by taking for comparison a familiar object, namely, a door which is kept closed by an elastic band. If such a door be pushed very slowly open, its pressure

against the hand will be greatest when it is most widely open, and will gradually diminish as it is allowed to close. If, however, it be pushed open quickly its acquired velocity will make it tend to leave the hand, and so diminish the pressure against it, and then as it recoils it will again come more firmly against the hand, and cause a second increase of pressure and a check to its rate of closing, or even an actual second opening. This effect is greater the greater the weight of the door in proportion to the elastic force, and would not occur at all if it were devoid of inertia. Similarly the effect of the inertia of the aorta will be that its contraction takes place with a spring-like recoil, which causes a second increase of fluid pressure, and this again produces a check to contraction or even a second expansion, which is one component of the dicrotic wave. Thus, so far as the dicrotic wave depends on this cause, it begins to occur in the aorta itself, but since the effect at any point is due to all the length of tube up to that point, it will be comparatively small in the aorta, and greater in the peripheral arteries, which is found in point of fact to be the case. The case of the aorta is, of course, very much more complex than that of the door, for the motion at any one point is closely dependent on that at every other point, and nothing can occur analogous to the pushing of the door quite away from the hand.

Thus the first cause of the dicrotic wave depends directly upon the inertia of the fluid, the second cause on that of the arterial walls in the first place, and only secondarily on the inertia of the fluid, because the motion of the tube and of the contained fluid must take place as a whole. The second cause simply tends to make the first arterial contraction greater than it otherwise would be, and to lead to its being followed by a second expansion. It will not, therefore, in general, give rise to a separate wave, but only enhance the dicrotic wave arising from the first cause. There can be no doubt on mechanical grounds that such an oscillation must theoretically take place; the only question could be whether it actually contributes to the dicrotic wave, or is too small to be noticed. If the motion at each point of the tube took place independently, the oscillation could be but small, because water is almost unyielding, but, as it is, yielding is allowed by the elasticity of the envelope in other parts.

The mechanism of these oscillations has some analogy to

what occurs when a stone is thrown into water. The first large wave which spreads in a circle around is followed by other smaller waves in a descending series. These are due, not to any reflection, but to the oscillation of the particles near the seat of disturbance. In experiments with an artificial heart and elastic tubes a similar descending series of waves is always obtained when the interval between the contractions is long enough, and it is not very uncommon, in a pulse which is very compressible but somewhat slow, to see, at an interval after the dicrotic wave not very much less than that between the primary and dicrotic, a repetition of the dicrotic, which I should propose to call the tricrotic wave. It is shown in Pl. I, figs. 2, 5, 26, and Pl. II, fig. 13. The slight wave, however, which often follows the dicrotic at only a short interval is due, like the tidal, to acquired velocity in the sphygmograph, and may be called the dicrotic-tidal wave (*vide* Pl. I, figs. 32, 34, 35, 37, and Pl. II, fig. 10).

I have introduced one tracing (Pl. I, fig. 29) to show that a pulse may sometimes be dicrotic, notwithstanding free aortic regurgitation. It was taken from a case of aortic and mitral disease, in which the pulse had generally the characteristic splashing quality, and sphygmographically had an appearance very opposite to that of the present tracing. For a short time, however, while the heart's action was very rapid and ineffective, it took the dicrotic form depicted. In at least one other case besides this I have noticed that, in a pulse of aortic regurgitation, dicrotism could be detected by the finger. It has been said by some that in such a case a double beat can indeed be felt, but that the two beats so felt are the percussion and tidal waves. In both these cases, however, the tidal wave was absent in the sphygmographic tracing, and the second beat that was felt followed the first at an interval corresponding to the dicrotic and not to the tidal wave. I do not believe that the primary and tidal are ever felt as two distinct waves, but I think that in some cases the finger may discern as separate phenomena two things which correspond in some degree to the primary and tidal waves in the tracing, namely, the first impulse of the artery against the finger, and afterwards its full expansion with blood.

In one or two patients in the last stage of mitral disease, in

whom the tricuspid valve had yielded, and a great backstroke in the veins of the neck occurred at each pulsation, I have observed that such backstroke was followed by a dicrotic wave in the veins which could easily be both seen and felt. This phenomenon could not be explained either by the common theory of the dicrotic wave or by that of Dr. Sanderson.

In order to be able to draw any mechanical inferences from the shape of the pulse curve, the most important point to determine is the cause of the variation of dicrotism. Since the recent conversion of an able writer on the subject, all observers are now fortunately agreed that dicrotism is increased by diminution of tension, and that thus, when it occurs in fever, it denotes a relaxation of the small arteries allowing a ready outflow of blood. Some, however, have supposed that it implies also a stagnation of blood in the veins. This could only be a direct cause if the dicrotic wave were due to a reflection from the periphery, which experiment disproves; and reason and observation combine in showing that the condition of the veins comes into play only in so far as it reacts upon the tension of the arteries. Any source of obstruction in the heart or lungs which causes increased fulness and elevated pressure in one part of the circle, namely, in the veins, must at the same time cause diminished fulness and lowered pressure in that part of the circle which is in front of the obstruction, namely, in the arteries. Such a kind of heart or lung disease increases dicrotism (*vide* Pl. I, figs. 26, 27, 30); but in the dicrotism of fever there is no sign of any venous stagnation, and the evidence points rather to diminished tension and increased rapidity of flow throughout the whole vascular system. So far as dicrotism depends on the state of the tubes in which it occurs, it is increased by an increase, not, in the strict mathematical sense of the word, of their elasticity, but of their distensibility. Thus, in the pulse of atheroma, combined with the increase of the tidal, there is a diminution of the dicrotic wave (*vide* Pl. I, fig. 4).

Observation shows that the dicrotism of fever and inflammation cannot be explained as being proportioned solely to the diminution of tension, for although, as a general rule, the most dicrotic pulses are the most compressible, yet there are some dicrotic pulses which will bear a pressure even greater than normal, and, again, there are some very compressible pulses

which are not very dicrotic. Some other element must, therefore, be sought in the causation of dicrotism, and I have endeavoured to show that this is to be found in the short and sudden action of the heart. Such a mode of action is in great measure merely the sequence of the less resistance it experiences owing to the lowered arterial tension and the freedom of outflow; but the want of constancy in the relation of dicrotism to lowered tension appears to show that it depends partly upon nervous influence affecting the heart directly. An instance is given in Pl. II, fig. 2, to show how a hyperdicrotic pulse like that of fever may be produced artificially without any elevation of temperature. It was obtained from a man aged 23, twenty minutes after a dose of four minims of nitrite of amyl, administered internally. The state of his pulse just before taking the drug is shown in Pl. II, fig. 1. The pulse thus obtained is somewhat intermediate in character between that of sthenic and that of asthenic fever, but approximates rather more nearly to the former. Judging by what is known of the physiological effects of nitrite of amyl, we may assume that the result was due mainly to the relaxation of the arteries, with probably, in addition, some degree of stimulation to the force of the heart's contractions. Notwithstanding the marked effect upon the pulse, the patient had himself perceived no effect whatever from the medicine.

Thus there are three things which tend to increase dicrotism, namely, diminution of tension, a jerky action of the heart, and distensibility of the arteries. It is probable that the dicrotism of fever may be in some degree promoted by a change in the last due to muscular relaxation, for although the dicrotic wave must be mainly produced in the largest arteries, in which the muscular element is small, yet the whole length of the artery takes part in some measure in the oscillation which is concerned in its production. Yet the presence of fever is not necessary for the production of great dicrotism, for I have never met with a dicrotic wave so large in proportion as in the pulse shown in Pl. I, fig. 26, which was obtained from a case of close constriction of the mitral valve. In that instance there was no fever, and the effect was due solely to lowered arterial tension, combined with the short sudden contractions of a dilated heart.

The state of things, then, which calls forth the greatest

dicrotism is a very low minimum tension in the arteries, which, at the moment of the heart's contraction, is suddenly, but briefly, raised to a maximum considerably exceeding that minimum, although generally in itself but small (*vide* Pl. I, figs. 3, 26, 29, and Pl. II, fig. 5). If the tension remain low, and the heart's action continue short, but become more feeble, the trace falls suddenly and deeply to the principal (or aortic) notch, but the dicrotic wave is of small absolute magnitude, although the pulse may be hyperdicrotic. Such a diminution of dicrotism often occurs in fevers after it has previously been considerable, and is then a sign of failing power. A pulse of this kind is very readily extinguished by pressure, and it is characterised by a loss of sharpness both of the top of the primary wave and of the bottom of the principal notch (*vide* Pl. II, figs. 6, 7, 9). If the rapidity of the pulse be at the same time increased, such a curve approaches to one of the forms which has been described as monocrotic, and which may occur in a very severe stage of fevers, such as typhus or typhoid (*vide* Pl. II, fig. 9). There is another form of pulse also called monocrotic, which is developed out of the ordinary hyperdicrotic pulse simply by increased rapidity, the dicrotic wave being lost upon the succeeding upstroke. This differs from the former by not indicating the same feebleness of heart's action, and hence it is not necessarily of the same bad prognosis, unless the points of the curve are at the same time rounded. I have never seen this state fully reached in the pulse of fever; but Pl. II, figs. 5 and 11, show a close approximation to it. The former was taken from a case of typhoid, very severe, but ending in recovery; the latter from a fatal case of typhus.

The kind of dicrotism produced by simply lowered tension, accompanied by such an alteration only in the heart's action as would follow secondarily from this, is perhaps best seen in a pulse taken immediately after an extensive loss of blood. Pl I, fig. 28, is a tracing obtained soon after a hæmorrhage from the lung to the extent of fifty-one ounces. It shows slight hyperdicrotism, a slanting primary upstroke, and considerable rounding of points, and it was very easily extinguished by pressure. A dicrotism much greater than this is often found a day or two after hæmorrhage, but this occurs when some degree of fever is superadded, as after wounds or surgical operations.

Pl. II, fig. 5, may suitably be compared with the tracing last mentioned, to show how that suddenness of the heart's action which exists in fever, when combined with a low tension, produces a dicrotism much greater than that which results from the latter alone, while at the same time the points of the curve are sharper. The tracing in question was taken after profuse hæmorrhage in a case of typhoid fever, and Pl. II, fig. 4, shows what the state of the pulse was, previous to the loss of blood. It will be seen that the amplitude was greater after the hæmorrhage than before; and since the diminution of tension by itself would tend to diminish amplitude by making the artery smaller, it would seem to follow that the effect of hæmorrhage was to cause a relaxation of the arteries even greater than that previously existing as a consequence of the fever.

One of the most important points as to the import of dicrotism is the question whether its increase is proportional to the elevation of temperature. The earlier writers on the sphygmograph considered that a close relation existed between the two. Thus it is stated by Wolff, that such a proportion is found, modified only by two circumstances, first, that a greater dicrotism belongs to chronic than to acute diseases at the same temperature; and secondly, that the pulse curve of old age, or that of aortic regurgitation, which he considers to be identical with it, goes through the same changes in fever as the healthy pulse, but at a slower rate, so that its dicrotism is less than that of the latter at the same temperature. Later observers, however, have found that the proportion between the increase of dicrotism and the elevation of temperature varies much in different diseases. I am inclined, however, to think that some have paid less attention than it deserves to the law of Wolff above quoted, in reference to the tardy appearance of dicrotism in senile pulses. It is to be remembered that, in reference to this quality in the pulse, age is not to be measured by years, but that the vascular changes which generally appear in advanced life may show themselves not unfrequently between the ages of thirty and forty, and even at an earlier period. Thus a non-dicrotic quality in a pulse associated with fever, which may at first sight appear due to a special character in the complaint, may really be the effect of pre-existing vascular degeneration. Hence, to find the pulse not dicrotic in a febrile disease such as pneumonia, in

which dicrotism generally occurs, may be of bad prognosis, especially if the patient be not very young, because it leads to the suspicion that his arterial system may be seriously damaged, and that he may therefore be less able to resist the effects of disease.

Again, the senile pulse curve, as it passes through the changes produced by fever, at a certain stage in its progress, assumes the form of the healthy pulse. Therefore, if a person be known to have very rigid arteries, to obtain from him a normal pulse curve may have the same significance as, in the case of a young person, to find a dicrotic pulse. Hence it is important that in all tracings which are to be used as evidence as to the state of the circulation in different diseases, the age of the patient should be noted, and that the temperature recorded should be taken at the same time as the tracing, or at an interval of not more than a few minutes, in order to avoid the errors which might arise from rapid changes of temperature. It is no doubt true that dicrotism is a quality which is more highly marked in the pulse of specific fevers, such as typhus or typhoid, than in that of other febrile conditions at an equivalent temperature. Yet I think that the difference is apt to be overestimated, from the fact that these fevers occur most commonly in young persons, while of cases of surgical fever or erysipelas a larger proportion are in persons who have reached or already passed middle life, and whose arterial system has no longer the elasticity of youth.

There is one kind of pulse with respect to which the sphygmograph might be expected to afford important evidence. I mean the incompressible bounding pulse of inflammatory fever, whose hardness used to be considered the warrant for bleeding. Of this, however, Dr. Sanderson declares that we have no opportunity of investigating it experimentally, since nowadays it is never met with. With this opinion I so far agree that I have never found a pulse which gave me reason to believe that the arterial tension had been increased by fever, understanding by the term arterial tension the tension in the intervals of the pulse, and not the maximum tension reached at the acme of the wave. It is true that it is not very uncommon to find associated with a high temperature a pulse which gives its greatest upstroke under a high pressure, and which also displays another, and perhaps more trustworthy, indication of high arterial tension,

namely, a considerable tidal wave. But in these cases the high tension is due not to the fever, but generally either to previous changes in the heart and arteries, to some morbid condition of the brain, or to albuminuria existing at the time. Such a pulse is shown in Pl. I, fig. 31. It was obtained not long before death from a fatal case of bronchitis in a man aged 60. In this case the temperature was not extremely high, being only 101·0° F., but the rapidity of the pulse was great, namely, 136 in the minute. The urine contained albumen, and the existence of a granular kidney was suspected, but after death the kidneys were found fairly healthy. The arteries were somewhat thick, but not atheromatous, and the heart was very slightly hypertrophied, weighing twelve ounces and a half. The peculiar state of the pulse in this case seems to have resulted from previous vascular changes of very moderate extent, coupled with the effect of a merely secondary and functional albuminuria in raising the arterial tension.

There is another sense, however, in which I do not agree with the statement of Dr. Sanderson before mentioned, for I think that we do now and then meet with pulses which to the finger have all the described qualities of the full hard pulse of sthenic inflammation, and I believe that the sphygmograph reveals the true state of circulation which then exists. The most typical example I have found of this is shown in Pl. I, fig. 3. That tracing was obtained from a strong ruddy-faced youth from the country, at the commencement of acute suppuration of the knee-joint, from a penetrating wound received two days before, and when his temperature was 104·5° F. The pulse bore a very high pressure, but it gave an even greater amplitude at a lower pressure, whence we must infer a very strong action of the heart, causing a high maximum arterial tension, but not a high minimum tension. The absence of the tidal wave shows the heart's contraction to be short although strong, and thus points to freedom of outflow from the arteries. The extent of dicrotism indicates a low minimum tension, combined with a sudden action of the heart, and the immense amplitude is partly the effect of the heart's vigour, partly of the dilatation of the radial artery. Thus there existed great relaxation of vessels, allowing a rapid outflow from arteries to veins, and consequently a low minimum arterial tension, but at the

same time a strong, sudden, but short action of the heart, which at each pulsation raised the tension to a high maximum. Both conditions together would contribute to accelerate the circulation in the highest possible degree. Pl. I, fig. 5, shows what the pulse of the same patient became after six days' illness. It had then become very compressible, and had all the qualities of asthenic fever.

Another pulse having similar characters, though not in quite such extreme form, is shown in Pl. I, fig. 7. The case was that of a man who the day previous had received a penetrating wound of the chest from a crowbar. The conditions present were acute inflammatory fever, with pleurisy and collapse of one lung, and the temperature was 104·8° F. The same combination is here indicated of low minimum arterial tension, with very powerful, though brief, contractions of the heart. The case was treated by bleeding to the extent of ten ounces. Fig. 8 shows the pulse immediately after the bleeding; fifteen minutes later some reaction had taken place, and the tracing then obtained is seen in fig. 10. Forty-five minutes after the bleeding the temperature had fallen four degrees, but it rose again to 103° F. before death, which took place the next day. It will be seen that this case forms an exception to the general rule that hæmorrhage increases dicrotism, for the pulse after the bleeding was somewhat less dicrotic than before. This is to be explained on the ground that the loss of blood being very moderate, its beneficial effect in lowering fever and diminishing the suddenness of the heart's action more than counterbalanced the tendency which it would otherwise have had, by diminishing tension, to increase the dicrotism of the pulse.

There are thus two varieties of hard pulse which may be associated with a high temperature, first, the dicrotic pulse, which bears a high pressure, as shown in the last two examples, which I regard as the typical pulse of very acute sthenic fever, apart from any cause leading to resistance in the circulation, and which denotes a ready outflow through dilated arteries, but, at the same time, a strong, sudden action of the heart. The asthenic dicrotic pulse differs from this in the fact that the vessels are still more dilated, but the heart's action less sudden and much less powerful. The second variety is the rapid pulse which is not dicrotic, and generally bears a pretty high pressure.

This, if not the result of previous arterial change, shows that the vessels have not undergone that relaxation which generally accompanies fever. If the amplitude of the pulse be at the same time very small, an actual contraction of the arteries must be inferred. Such a pulse is found especially in peritonitis, and is recognised by the finger as the small hard wiry pulse.

I shall now proceed to notice in succession the kinds of pulse found in several kinds of acute disease and the state of circulation to be inferred from them.

1. *Surgical Fever.*

It has already been shown that sthenic surgical fever in a young person may produce a very dicrotic pulse, yet its tendency to dicrotism is less than that of typhus or typhoid fever. Thus at moderate temperatures the pulse may remain non-dicrotic, and if there be in addition some impairment of arterial elasticity, or if albuminuria be present, this may be the case even at a very high temperature. Some degree of vascular change may have commenced in a man whose pulse is shown in Pl. I, fig. 9, but no apparent atheromatous condition was found post-mortem. The patient had undergone an operation about the tongue, followed by so much hæmorrhage that a ligature was placed upon the common carotid artery two days before the tracing was taken. This was followed by hemiplegia, and the temperature had risen to 104·7° F. at the time of procuring the tracing. An inspection afterwards showed that cerebral softening had occurred. Thus, in this case, besides the fever, there had been at work another cause which generally tends to dicrotism, namely, the loss of blood. The pulse, however, though very quick, is but little dicrotic, and of small amplitude. It will be shown afterwards that this form of curve, which is like that of peritonitis, is commonly found in the rapid pulse of cerebral disease. It is probable, therefore, that it may have been due, in this case, to the condition of the brain, and, if so, must imply that irritation was transmitted to the vaso-motor nerves, unless it be held that, as in the case of the heart, so in that of the arterial system, a paralysis of cerebro-spinal centres may produce the same effect as irritation of the sympathetic.

When surgical fever becomes more chronic the pulse soon

becomes compressible, and assumes the asthenic dicrotic form. An example of this is shown in Pl. I, fig. 5, and also in Pl. I, fig. 2. The latter was obtained from a girl aged 21, on whom the operation of excision of the knee-joint had been performed six days before on account of chronic disease. The pressure is low, the dicrotic wave large in proportion, the points of the curve somewhat rounded, and the tricrotic wave is also seen, as it is also in fig. 5. In Pl. I, fig 6, is shown a pulse which approximates more nearly to the asthenic than to the sthenic type, although it was taken quite at the commencement of acute suppuration of the bursa patellæ in a man previously healthy, whose temperature at the time was 104·6° F.

2. *Erysipelas.*

The pulse in erysipelas has much resemblance to that in surgical fever. When an acute case occurs in a young person, the kind of pulse found at an early stage is generally the sthenic form of dicrotic pulse, which bears a somewhat high pressure and has considerable amplitude. An example of this is the tracing shown in Pl. I, fig. 11, which was obtained on the second day of acute medical erysipelas of the face, when the temperature was 104·5° F. Sometimes, even in young persons, the curve is more allied to the non-dicrotic form of hard pulse, as in the case shown in Pl. I, fig. 12, in which the presence of the tidal wave and a dicrotism which is scarcely full, shows that comparatively little vascular relaxation could have occurred, although the temperature was 105·5° F. Such cases are peculiarly suited for treatment by aconite and other remedies of the same nature.

The pulse of sthenic surgical erysipelas is of a similar character to that of medical, but if it assumes an adynamic quality the pulse becomes compressible, and takes the asthenic dicrotic form, with rounded points to the curve, and a greater dicrotism in proportion to the temperature. As is the case with surgical fever, if erysipelas occur in a person not very young, in whom vascular changes may have commenced, the pulse often shows no increase of dicrotism at all, and may retain evidence of high minimum tension in the presence of a considerable tidal wave. In Pl. I, fig. 13, is shown a tracing obtained on the fourth day

of a case of erysipelas of the face in a man aged 56, whose arteries felt rigid, and whose temperature at the time was 103° F. In fig. 14 is shown the pulse of the same patient twelve hours later, during which time he had taken twenty-five minims of tincture of aconite. The pulse has become more dicrotic, and presents all the signs of diminished arterial tension. At the same time the patient was sweating profusely, his delirium had subsided, and his temperature had fallen to 99·8° F. It is thus evident that although the considerable arterial tension which in this case was at first associated with high fever, might be partly the result of degenerated vessels, it yet must have been partly due to the nature of the disease, since the character of the pulse was so rapidly changed by the action of the remedy, for what could be effected by drugs might also have been effected by the fever.

In Pl. I, fig. 15, is shown the pulse of a woman aged 29, in whom there was no reason to suspect any arterial degeneration. In this the presence of the tidal wave, the high tension, and the absence of any increased dicrotism, combined with the small amplitude, appear to denote a general contraction of arteries, and, therefore, a condition exactly the opposite to that relaxation which generally exists in fever. The case was one of surgical erysipelas, after an operation about the vulva, and it was accompanied by extreme pain. It appeared possible that the effect of this upon the nervous system might be to produce a state of vessels similar to that which exists at the initial stage of fever, when rigors occur, and often in inflammations of serous membranes, in both of which cases the pulse is non-dicrotic. It is to be noted, however, that in this case the temperature was not very high, being only 101·5° F.

3. *Acute Rheumatism.*

In this complaint I have found dicrotism to be less than in any other acute febrile disease. This appears to be partly because the range of temperature is not in general excessively high, and partly because it shares with surgical fever and erysipelas the peculiarity of having less tendency to produce dicrotism than is found in most zymotic fevers. Thus, in the milder degrees of fever, a fairly-developed tidal wave may indi-

cate that the tension is high and that there is no vascular relaxation. Yet it is not uncommon to find a nearly full dicrotism, especially in feeble persons or in the later stage of the complaint, if the fever continues high. When the hand is affected on the side where the tracing is taken the amplitude is very large, and the dicrotism is generally somewhat greater, while the pulse still bears a rather high pressure. Thus the pulse changes somewhat from the sthenic non-dicrotic towards what I have called the sthenic dicrotic type, in consequence of the local inflammation and the active flux which accompanies it. This confirms the view that such a form of pulse denotes increased freedom of outflow, but an undiminished or enhanced vigour of heart's action. The increased amplitude depends on dilatation of the radial artery, and the increased dicrotism is probably due also to vascular dilatation in the affected limb, since the whole length of artery between the heart and the point where the tracing is taken has some share in the production of the dicrotic wave, and dicrotism increases with an increase in the distensibility of the tube in which it is produced.

The common form of pulse in acute rheumatism is shown in Pl. I, fig. 16. The tracing in fig. 17, taken from a case in which the temperature was lower than in the preceding, shows how greatly the tendency to dicrotism is increased by the complication of mitral regurgitation, on account of the influence which that has in diminishing the arterial tension. I have more than once had my attention attracted by a change of this sort in the pulse to the commencement of endocarditis, when it came on during the course of the disease.

4. *Peritonitis.*

The pulse of peritonitis has been often described, and its sphymographic characters agree with those discovered by the finger. It is of small amplitude, rapid, and non-dicrotic, or but little dicrotic, even when the temperature rises to a high point (Pl. I, figs. 18, 21). Thus the state of circulation indicated is exactly the opposite to that which generally occurs in fever, except as to the rapidity of the pulse. The radial artery is contracted, and there is no freedom of outflow, from which it is to be inferred that the small arteries are contracted

also. The heart, however, is not stimulated by this to strong slow contractions as under normal circumstances it would be, but runs on with short feeble beats. The same kind of pulse, although not in so marked a degree, may be found in acute pleurisy, and in pericarditis, and it also occurs in colic, in the cold stage of ague, and during the rigors which usher in a febrile attack. It would seem that there must be one state of the nervous system common to all these conditions, namely, irritation of the vaso-motor nerves; and the fact that this is more greatly developed in peritonitis than in any of the rest may be accounted for on the ground that the sympathetic nerves are involved over a very wide surface in that disease. Irritation of the sympathetic may also account for the fact that the rapidity of the heart's action departs so widely from the relation by which, as shown by M. Marey, it is generally connected with the vascular tension, an occurrence which must indicate that its innervation is profoundly affected.

The pulse of peritonitis generally bears a pretty good pressure, but it may become at length compressible while still remaining non-dicrotic. In that case the absence of dicrotism must be due to the feebleness of heart action rather than to high arterial tension. A dicrotic pulse may sometimes occur when peritonitis exists, if other conditions are superadded. An example of this is shown in Pl. I, fig. 19, a tracing obtained from a woman aged 31, on whom the operation of ovariotomy had been performed five days before. In that case there was suppuration about the wound, and the patient was in a very low state, for the tracing was obtained only a few hours before death.

The tracing in Pl. I, fig. 22, was obtained from a woman aged 26, who, twenty-five days after the operation of ovariotomy was attacked by severe vomiting, and other symptoms somewhat resembling peritonitis. The pulse is most unlike that of peritonitis, the pressure borne is a very low one, and both dicrotic and tricrotic waves are well marked. In this case an inspection revealed that there was hardly any peritonitis, but a rapid diffusion of malignant growth.

5. *Pericarditis.*

When slight pericarditis occurs in the course of acute rheumatism, no characteristic alteration recurs in the pulse; if, however, it be more considerable, there is generally an increase in the rapidity of the pulse without any corresponding increase of dicrotism. In those cases, however, in which severe pericarditis produces a marked effect on the pulse, the typical form of curve that results is not unlike that of peritonitis, except that it is not always of such small amplitude, for it is rapid without being dicrotic (*vide* Pl. I, fig. 23). In some cases the heart may be thrown into extreme perturbation, as shown in Pl. I, fig. 24. That tracing was obtained from a case of pericarditis set up by pyæmic abscess of the heart. The larger elevations seen are due to the respiratory curve, the smaller notches are the pulsations, which were at the rate of about 360 per minute.

There are cases in which the pulse of even severe pericarditis shows considerable dicrotism, as in Pl. I, fig. 25. In that case, as so often happens, when convalescence had commenced and the pericardial sounds had disappeared, an endocardial bruit became audible. It appeared probable, therefore, that the dicrotic quality of the pulse might have been due to the commencement of mitral regurgitation, which did not until some time afterwards manifest itself by a bruit.

6. *Pneumonia.*

In its effect on the pulse pneumonia occupies a somewhat intermediate position between the three diseases first noticed, namely, sthenic surgical fever, erysipelas, and acute rheumatism, on the one side, and the zymotic fevers, as typhus and typhoid, on the other. As a rule the pulse in pneumonia is dicrotic, and I have not met with exceptions to this rule under the age of twenty-five. Sometimes even a considerable degree of hyperdicrotism is reached (*vide* Pl. II, fig. 15), but yet the dicrotism is not quite so great in proportion to the temperature, at any rate in the milder degrees of fever, as it is in typhoid. The pulse also differs from that of typhoid in two other respects—

first, that it bears a higher pressure, at least in the earlier stages of the complaint; and, secondly, that the points of the curve are sharper, and the upstroke more nearly vertical. Hence of the two causes of dicrotism, namely, lowered tension and suddenness of heart action, the latter plays comparatively a greater part in pneumonia than it does in typhoid, and the pulse approximates somewhat more nearly to the sthenic dicrotic type. When, however, the pneumonia occurs secondarily in the course of phthisis, the pulse may take at once the asthenic dicrotic form (*vide* Pl. II, fig. 18). If in pneumonia occurring in a person who has reached middle age the pulse be found non-dicrotic, while at the same time the temperature is high, and other signs of severity exist, the absence of dicrotism must be considered rather an unfavorable sign, because it then indicates, not that the fever is mild, but that the arterial system has undergone degeneration, or else it is associated with the presence of albuminuria. An instance of this is shown in Pl. II, fig. 17. It was obtained from a case of pneumonia in a man aged 54, in which albuminuria was present, and which eventually proved fatal, and was taken when the temperature was 102·2° F. Secondary albuminuria, however, in pneumonia or in typhoid fever may often coexist with a dicrotic pulse, for although it appears to have a tendency to increase arterial tension, due either to the state of the kidney, or to the state of blood antecedent to the albuminuria, yet this may be counterbalanced by the other conditions present.

7. *Typhus and Typhoid Fever.*

It is in the case of these fevers that the indications of the sphygmograph relatively to prognosis have been already most fully and most usefully treated by previous writers. I shall here only speak of them in reference to the conclusions which must be drawn in respect to the state of the circulation which exists in them. At the outset of typhoid fever in strong persons the pulse, while quickly becoming dicrotic, may for a time bear a moderately high pressure, and thus have something of the quality of the sthenic dicrotic type. Soon, however, and it may be from the commencement, it becomes very compressible, although at quite a low pressure the amplitude may be consider-

able. The upstroke also grows somewhat slanting, and both the summits and the bottom of the principal notch become rounded, so that when the fever is severe the line of the tracing presents an undulatory appearance (*vide* Pl. II, figs. 7, 8). Typhus fever differs from typhoid only in the fact that the pulse more rapidly assumes the asthenic quality, and shows it in a higher degree (*vide* Pl. II, fig. 11). Everything combines to show that in this case the principal element in the causation of the dicrotism is the extreme lowness of the arterial tension; and we must infer that in these fevers more than in any other disease a relaxation of the vessels takes place, which allows a ready outflow from the arteries into the veins. The heart, however, is acting with much less suddenness and power than in the more sthenic forms of fever, and it appears probable that the actual rapidity of the circulation is much less than that which may be attained in such cases when the heart is vigorous and the vessels at the same time moderately relaxed.

In two cases at the commencement of typhoid I have found a pulse-curve of a shape which I have not seen noticed by any writer on the subject; it is shown in Pl. II, fig. 3. The pressure is not very low, the primary summit is rather pointed, and the trace falls deeply from that point to the principal notch, which is sharp and is followed by two secondary waves. From this must be inferred an arterial tension not quite so low as commonly happens in typhoid, and a contraction of the heart, which is sharp at the commencement, but shorter than is usual, even in fever. The result is that the trace rises to a summit more above the level of the true pulse-wave than is generally the case in dicrotic pulses. From this point it falls rapidly, but does not catch the still more rapidly falling true wave until the bottom of the principal notch is reached, from which it rises to a wave preceding the dicrotic wave, and in this case due solely to a recoil in the sphygmograph. Therefore, although corresponding to the tidal wave, it no longer indicates the passage of the arterial tide, and it might, therefore, be called the pseudo-tidal wave. The resulting curve thus looks like a pulse in which the dicrotic wave has become reduplicated. A curve somewhat similar is occasionally found in one form of the rapid pulse of cerebral disease. The cases of typhoid in which I observed such a pulse-curve ran afterwards a very severe course,

and were accompanied by much delirium. This form of curve, however, only lasted for a day or two, and was then replaced by the ordinary dicrotic pulse. Somewhat similar forms of curve, obtained in cases of cerebral disturbance, are shown in Pl. I, fig. 20, and in Pl. II, fig. 27. Another case, in which the dicrotic wave is also sometimes broken into two by the effect of recoil in the sphygmograph, is when fever supervenes in a person whose heart has previously become hypertrophied, and makes his pulse dicrotic. The hypertrophied heart then appears to adopt a mode of contraction somewhat resembling that which, in the other case, is the result of nerve irritation.

A form of pulse which is very important in its bearing on the question of what is the true physiological condition in fever is that which sometimes occurs in typhus or typhoid, and is fully developed only when the disease is approaching a fatal termination (*vide* Pl. II, fig. 9). The primary summit becomes broadly rounded, and may even approach to a square shape, while, at the same time, the dicrotic wave becomes smaller in proportion, though the principal notch is even more rounded than before. Dr. Anstie has specially noticed the very unfavorable prognosis to be derived from a pulse like this, and he explains it as denoting a contracted state of the small arteries. If this be so, then we must suppose that, in this most severe stage of fever, there supervenes a state of vessels precisely the opposite of that which is characteristic of fever in general, and especially of these particular fevers. Dr. Anstie has not recorded the pressures at which his tracings were taken, but I have generally found that when the pulse underwent this ominous change it became even more compressible than before. I should, therefore, explain it as indicating a more feeble and sluggish, though quickly repeated action of the heart, which fails to call out any considerable dicrotic wave, although the arterial tension remains as low as before. I consider, therefore, that the state of circulation in typhus or typhoid differs from that which exists in most other forms of fever in the fact that, while the vascular tension is still lower than in them, the heart's action is more feeble and sluggish, and that it is simply the exaggeration of the latter quality which tends to the development of this kind of monocrotism.

It is evident that the same sluggishness of recoil which, when tension is high, can only be due to a long vigorous action of the

heart, the consequence of obstructed outflow or impaired arterial elasticity, may, when tension is very low, result from the extreme feebleness of the distending force. Thus, the feeble pulse of mitral disease (Pl. I, fig. 30) shows a roundness of summits very like that of the undulatory pulse of fever (Pl. II, fig. 8). It is true that the diminution of the dicrotic wave does not occur when the dilated heart of mitral disease is beginning to fail. It may, indeed, be somewhat difficult to tell whether such a pulse is absolutely monocrotic, or whether every second elevation is the dicrotic wave, but I believe that the latter is the true explanation (Pl. I, fig. 30). The different state of things in the pulse of fever may be explained partly on the ground that in that case the rapidity is greater in proportion to the magnitude of each beat. Thus, in the almost monocrotic pulse of fever, a late dicrotic wave is partly cut off by the succeeding upstroke. On the other hand, if the mitral pulse becomes very rapid it becomes also very small, and thus the dicrotic wave, following more closely, finds room for itself before the next upstroke, as in Plate I, fig. 30. Another reason, probably, is that the action of a dilated heart has a peculiar shortness, which is specially adapted for calling out the dicrotic wave (*vide* Pl. I, fig. 26). I have, however, met with a few cases in which the sphygmograph appeared to give evidence of such an obstructed circulation as that described by Dr. Anstie. One of these is shown in Pl. II, fig. 12, which was obtained from a fatal case of typhus in which the pulse had before shown the ordinary dicrotic character. The pressure is somewhat greater than it had been previously. The primary wave is very broad, and not only is the dicrotic wave little marked, but there is no deep fall preceding it. The arterial tension must, therefore, have become somewhat elevated in the later stage of the fever at the same time that the heart became sluggish. In the cases of this kind which I have observed the urine has been albuminous, and the effect is probably to be attributed to a state of blood either the result of the condition of the kidneys or itself a consequence of the fever, and antecedent in point of causation to the albuminuria. But the effect of the fever-poison in itself, by its action, as it would seem, on the nerve centres, must be to cause vascular relaxation, and not contraction, since there is evidence that in these fevers such relaxation occurs in a greater proportion

than usual to the elevation of temperature. Thus, in this rare case of obstructed circulation in fever, we are driven for explanation to the same alternative as in the case of that obstruction which is the rule in Bright's disease even when acute,—either that the altered blood is delayed in its circulation by a modification of capillary forces, or that it causes a contraction of the arterioles by means of reflex irritation conveyed through the peripheral nerves. Of these two explanations the former would avoid the necessity of supposing that the same poison is capable of producing, through the nervous system, two opposite effects.

There is a form of febrile complaint, not uncommon, in which a person is attacked suddenly by feelings of severe malaise, and the temperature rises rapidly to a high point, such as 103° or 104° F. Such an indication of the thermometer is apt to cause an impression that some severe disease must be commencing, and even to raise a suspicion that the complaint may be typhus fever. In a few days, however, it all passes off, and the attack has to be reckoned as being nothing more than febricula. It is a malady which is very commonly observed in hospital nurses. In one or two cases of this kind I have found that, notwithstanding the high temperature, the pulse-curve had a shape very different from that of typhus, and, indeed, not deviating widely from the standard of health. One of these is shown in Pl. II, fig. 14. Further experience, however, would be required to show whether the sphygmograph can be used in such cases as affording a more reliable aid to diagnosis than the thermometer.

8. *Cerebral Disease.*

Among the obvious qualities of the pulse in cerebral disease the chief thing to be noted is that the most opposite varieties may be found in different cases—in one a very slow pulse and in another a very rapid one, and that it is often impossible to determine the precise reason of the difference. Moreover, the rhythm may be irregular, and in this way the pulse may change from one type to another within very short intervals. The sphygmograph, while confirming these facts, adds another of a similar character, namely, that the rapid form of cerebral pulse may in different cases present curves of quite opposite character, sometimes dicrotic and sometimes the reverse, sometimes of high

tension and sometimes of low. These different forms of pulse must, no doubt, severally correspond to particular states of the nerve centres, but what those states are there seem to be at present no means of ascertaining.

There is one form, however, which presents considerable constancy of character, and that is the slow variety of cerebral pulse, such as often occurs from compression of the brain, or as a consequence of a blow upon the head; such a pulse is shown in Pl. II, fig. 19 and fig. 20. The pressure is considerably higher than normal, and the curve sustained. The primary wave is well marked, but its upstroke often somewhat slanting, as if in consequence of the high tension. The tidal wave is large and expanded, denoting a prolonged flow of blood. Now the slowness of the heart's action must by itself tend to lower the arterial tension, since the quantity of blood pumped into the arteries is thereby rendered smaller. Therefore the increased tension in the slow cerebral pulse shows that the slowness of the heart's action is more than counterbalanced in its effect upon the arterial tension by a contraction of the arterioles, which diminishes the freedom of outflow. It may be concluded that the mode of action of the heart is, in the main, secondary to the increase of arterial pressure, and that the slow cerebral pulse is therefore not due to direct nervous influence acting on the heart so much as to an excessive action of the vaso-motor nerves throughout the body from irritation communicated to them.

This form of pulse may be usefully compared with the slow pulse produced in a healthy person by the application of external cold (*vide* Pl. II, fig. 21), the effect of which will be generally allowed to be a contraction of all the vessels. The curve in this case differs only from the former one in the fact that the amplitude is less, and the several waves less distinctly separated, owing to the "percussion element" being smaller. Both these circumstances point to less vigour in the heart's contraction. In the cerebral pulse shown in Pl. II, fig. 19, the curve appears to show an irritable suddenness in the commencement of the contraction, and its form approximates somewhat to that of Bright's disease occurring in a young person. But in a more severe form of cerebral affection, when coma becomes deep, the heart's action loses its suddenness and force, and the pulse-curve first takes a form like that of the pulse produced by cold (Pl. II,

fig. 21), and afterwards becomes of yet smaller amplitude, though still sustained and bearing a somewhat high pressure.

The rapid cerebral pulse is, as a rule, not dicrotic, and requires for its development a pretty high pressure, and it thus affords an instance in which the rapidity of the pulse is not secondary to the lowering of arterial tension, but must depend upon nervous influence acting directly upon the heart. This is the usual form of curve when the pulse becomes rapid in coma, as shown in Pl. II, fig. 23 and fig. 26. In the latter case there was but little dicrotism, although the temperature had reached so high a point as 105·7° F. The case was one in which coma and death followed a series of epileptiform fits repeated at short intervals. There was no albuminuria, and no lesion of brain was found post-mortem. The pulse has a small amplitude like that of peritonitis and its tension is not very low; whence it must be inferred that, notwithstanding the very high temperature, there was an excessive action of the vaso-motor nerves, producing a general arterial contraction.

If the rapid cerebral pulse becomes dicrotic, it belongs, as a rule, to the sthenic dicrotic type; the points of the curve are sharp, the pressure not very low, and the dicrotism not very great in proportion to the temperature. Two instances of this are shown in Pl. II, fig. 30 and fig. 31. The first was obtained from a woman aged 30, who was attacked by tubercular meningitis during the course of phthisis. This tracing was taken when she had become comatose, and it will be seen that full dicrotism had just been reached at a temperature of 103·2° F. In the earlier stages of the cerebral disease the dicrotism was scarcely more than normal, and the pulse presented a contrast to its previous condition, which resembled that which often occurs in phthisis when the temperature is elevated, namely, one approximating to the asthenic, undulatory dicrotic type. The second case was one of extensive sclerosis of the brain and spinal cord in a woman aged 23. After the disease had long been running a very chronic course the temperature suddenly began to rise rapidly, the patient became comatose, with lividity of face and oppressed breathing, and died in a very few hours. The tracing shown in Pl. II, fig. 31, was taken about two hours before death.

Another form of rapid cerebral pulse is that shown in Pl. I,

fig. 20, which was obtained from a case of fatal cerebral rheumatism in a woman aged 40, after profound coma had commenced, and the temperature had risen to 106·2° F. The dicrotism is not excessively great, thus denoting that vascular relaxation had not progressed at the usual rate in proportion to the increase of temperature. The form of curve somewhat resembles the unusual form found at the outset of typhoid (*vide* Pl. II, fig. 3), and, as in that case, indicates an irritably sudden and short heart's action, with a vascular tension not very low.

In all these cases the arterial relaxation was either entirely absent, or its degree was less than usual, in proportion to the increase of temperature. In Pl. II, fig. 24, however, is shown a pulse in which the dicrotic wave has a very large proportionate magnitude, and the pressure is very low, while the elevation of temperature is only moderate (101·6° F). The case was one of traumatic meningitis, secondary to comminuted fracture of the skull, and exposure of the brain substance. Soon after the accident, and four days previously to the time at which this tracing was taken, the pulse had shown, in a marked degree, the very opposite state of circulation, namely, a much greater elevation of temperature (103·5° F.), with but little, if any, vascular relaxation, and apparently an actual increase of arterial tension (*vide* Pl. II, fig. 22). The dicrotism is but little more than normal, the tidal wave still appears, and the pressure is as high as five ounces. It is evident that the arterial pressure may be actually increased, notwithstanding some degree of vascular relaxation, if at the same time the heart is acting more rapidly than usual, and not ineffectively, and this may have been the case in the pulse in question.

There is one cerebral condition in which there is commonly a rapid pulse without any increase at all of temperature, and that is in acute mania. It will be readily understood that in this case, when excitement is great, the obtaining a tracing is a process of no little difficulty, and one which may involve great peril to the sphygmograph, while in the more chronic forms of mania the pulse often presents nothing of interest. In the case of mania the variability in the degree of dicrotism, which is associated with the same rapidity of pulse, is still more marked than in other forms of cerebral disease. The pulse, which at one time is hyperdicrotic, may in the same person at another

time show a normally shaped curve, while still remaining as rapid as before; and sometimes, while the sphygmograph remains fixed upon the arm, a change in the degree of dicrotism may be seen in accordance with the rapidly changing emotions. In Pl. II, fig. *32*, is shown one case in which the pulse was hyperdicrotic. In this instance, therefore, the state of circulation generally accompanying fever, and of which the most important element is vascular relaxation, is found to coexist with a normal temperature. It has already been shown that the same state of things may be produced by the administration of nitrite of amyl. In the present case, however, the points of the curve are sharper, and the dicrotic wave shows a tendency to be broken into two by the effect of recoil in the sphygmograph, thus indicating more of irritable suddenness in the heart's action. From this fact, that a febrile pulse may coexist with a normal temperature, it may be inferred that the excess of heat in fever is not the effect of the increased supply of blood to the tissues, nor in any other way the result of the state of the vascular system which generally accompanies it. But the association of the two is so general, whatever be the origin of the febrile state, that there must be some link of causation between them, and it may therefore be concluded that elevation of temperature tends to cause vascular relaxation, although this effect may be modified or even quite counteracted by other conditions.

A very dicrotic pulse, however, is the exception in mania, and a more common form is that shown in Pl. II, fig. 29, a tracing obtained on a different occasion from the same patient. In this curve the tidal wave appears, and while the amplitude is small the pressure required is unusually great. Thus, on this occasion there was an abnormal contraction instead of abnormal relaxation of the vessels. Another specimen of the pulse of mania is shown in Pl. II, fig. 25. Although a rapid pulse obtained from a girl of 20, its shape resembles that of the slow pulse of old age, for the tidal wave forms a convexity following close upon the primary summit, while the pressure is greater than normal. This form of curve, if not the effect of arterial degeneration, which would seem unlikely in so young a person, must indicate that, while the outflow from the arteries is obstructed, and the arterial tension high, each

contraction of the heart is prolonged, although the beats are quickly repeated. This pulse must be contrasted with that of peritonitis and other similar conditions (*vide* Pl. I, fig. 18), in which there exists the same vascular contraction and consequent increase of arterial tension, but the heart's action, while becoming rapid, becomes also short, so that the tidal wave is not increased, but generally diminished or lost. Thus in this case of mania the effect on the arteries was similar to that produced by irritation of the sympathetic system, but that on the heart was different.

In Pl. II, fig. 28, is shown a tracing obtained from a case of subacute mania in a woman aged 25, whose pulse was not very rapid. It differs from a normal pulse in that the pressure is greater and the tidal wave more prolonged; the amplitude is also rather small. Hence in this case also there was arterial contraction and prolonged action of the heart.

Thus we see that in cerebral affections, including mania, there are found greater abnormalities than occur in any other diseases as regards the relation which generally subsists between the temperature, the rate of pulse, and the relaxation or contraction of the vessels. As a general rule there is less vascular relaxation than usual in proportion to the increase of temperature. Examples, however, are found of the opposite extremes in both directions; on the one side great vascular relaxation, with a normal temperature, and, on the other side, and more commonly, a very high temperature combined with actual vascular contraction, instead of any relaxation—a state of things which may occur also when there is irritation of the sympathetic, as in the case of peritonitis. We must conclude, therefore, that in diseases of the brain there is generally a transmission of irritation to the vaso-motor nerves, unless it be supposed that, as in the case of the heart, so in that of the arteries, besides the centres of stimulation, there are other centres of inhibitory action, whose locality has yet to be discovered, and that these may become paralysed in some cerebral affections. It is certain that in some cases, such as that of severe concussion, the occurrence of paralysis would seem more probable than that of over-action.

9. *Acute Nephritis.*

The state of the pulse in chronic Bright's disease as displayed by the sphygmograph has been discussed by Dr. Sanderson and several other writers. Its characters are high pressure, generally a large amplitude, a tidal wave which occurs early and is broadly expanded, and a dicrotic wave rather small in proportion (*vide* Pl. I, fig. 33). All these testify to an obstructed outflow, causing high arterial tension and a prolonged vigorous action of the heart.

In many cases of tubal nephritis occurring in persons who have reached middle age I have found that the pulse shows considerable approximation in character to that typical form which belongs especially to a granular kidney. In such cases, however, it is very difficult to be quite certain that an insidious chronic change had not preceded the acute attack; but that Bright's disease in itself does exert a marked influence on the pulse is shown by the effect of acute nephritis in young persons, for in such cases a most characteristic form of tracing is often found. Such a pulse-curve in its most pronounced shape occurs in no other condition, and recently, out of five consecutive cases of acute nephritis of from three weeks' to three months' standing, in patients under the age of twenty-five, I have found it typically shown in every one. There are some cases, however, in which it is absent, just as in some of granular kidney the characteristic form of pulse is sought for in vain.

Four of the tracings referred to are shown in Pl. I, fig. 32, fig. 34, fig. 35, and fig. 37. The pressure is somewhat above normal, but the excess is not so great as in cases of granular kidney in old persons, and the amplitude of the trace is generally rather less than usual. The tidal wave is strongly marked, as in the case of Bright's disease in an older person, but its shape is different. Instead of being broadly rounded it has a rather pointed summit, which may rise nearly as high as the primary wave, and it is preceded by a somewhat deep notch. So different is its aspect that, on first seeing such a trace, one would be apt to consider that it could not be the same wave as the tidal wave of the form shown in Pl. I, fig. 4 or fig. 33, but their identity is established by the fact that all intermediate steps between the

two shapes may be found. This peculiarity of shape shows that the tidal wave does not in this case simply coincide with the corresponding part of the true pulse-wave, and so denote only the prolongation of the arterial tide, but that it is partly due to the element of recoil, which is brought into play by the fact that, the primary summit having risen considerably above the true wave, the first notch dips in its turn below it. A second oscillation of the same kind adds somewhat to the height of the dicrotic wave, and third, and perhaps even a fourth, may follow that wave.

The actual features, then, which are seen in these peculiar curves are due in part to an oscillation set up in the sphygmograph, but the pulse must have some very peculiar quality to produce this effect, since it occurs under no other circumstances; and no doubt its peculiarity is made much more manifest in this way than if the actual form of the true wave could be quite literally transcribed. The quality to which the result is due must be an extreme suddenness in the commencement of the wave, which, however, is, at the same time, well sustained, as is proved by the development of the tidal wave; for if the heart's action were jerky, and not, at the same time, prolonged, the trace would fall suddenly without any tidal wave, and form a dicrotic curve, as it may do when fever is superadded to Bright's disease (*vide* Pl. I, fig. 36). Thus, in acute nephritis as well as in chronic, there is commonly an obstructed outflow from the arteries and consequent elevation of tension. The small amplitude of the trace might at first sight lead to the conclusion that the theory of Dr. Johnson, that the small arteries are spasmodically contracted in Bright's disease, is true, and that such contraction occurs not only in minute vessels, but in arteries of the size of the radial. It is open, however, to another explanation, for if the arterial tension be raised and the heart's contraction be thereby protracted, the pulse-wave, while becoming longer, will be diminished in height, and the amplitude of the trace will therefore be less. It will be seen that in one tracing, obtained from a patient in whom the disease had lasted three months, the amplitude is considerable, as if some hypertrophy of the heart had already taken place. It is evident that arterial contraction alone would not account for the whole effect, for in that case the curve would be the same as that produced

by cold (*vide* Pl. II, fig. 21), or by the use of ergot, but it is very different.

In several cases in which symptoms of uræmia had appeared in young persons without any elevation of temperature, I have found the amplitude still smaller, and the pressure required to develop the pulse extremely high, namely, from six to ten ounces. In these cases, therefore, with the appearance of the uræmic symptoms, the signs of obtruction to the circulation became greatly enhanced, and it might be suggested that some, at least, of the cerebral symptoms in cases of the kind are due to the deficient blood supply which would result from such obstruction. But, on the other hand, it is possible that there may be an arterial contraction which is itself secondary to the state of the brain produced by uræmia, since it has been shown that such contraction frequently accompanies cerebral disturbance.

In cases of Bright's disease in young persons, in which inspection afterwards shows the kidney to be small and granular, the form of pulse-curve closely resembles that which was found in these instances of acute nephritis, including even the smallness of amplitude. This I have found to hold true in the case of two patients under the age of twenty, in whom such a kidney was found, and whose hearts were hypertrophied, weighing as much as seventeen ounces. In them, therefore, the increase of vascular tension, if not an actual contraction of the radial artery, must have more than counterbalanced the effect of the heart hypertrophy as tending to increase the amplitude of the pulse.

The evidence of the sphygmograph, therefore, is that in albuminuria there is generally an obstructed outflow and consequently elevated arterial tension, whether this be due to arterial contraction or to a physiological impediment to the circulation of the blood through the minute vessels, which results from a change in its relations to the tissues, and a consequent modification of capillary forces. But if an acute inflammation arise in the course of Bright's disease, and the temperature be thereby considerably raised, the pulse may become dicrotic; or, in other words, the effect of fever in relaxing the vessels and thereby lowering tension overcomes the tendency of the Bright's disease to increase tension by causing the circulation to be impeded. The heart, however, still retains

some of its vigorous suddenness of action, so that the points of the curve are sharp, the dicrotism not very great in proportion to the temperature, and the dicrotic wave often shows a tendency to be broken into two from the effect of recoil. This character is all the more marked if the Bright's disease be chronic, and the heart, therefore, have previously become hypertrophied. Two instances of a dicrotic pulse in Bright's disease are shown in Pl. I, fig. 36, and Pl. II, fig. 27. The first was obtained from a case of pleurisy and purulent pericarditis in a girl aged 18, who had previously been one month ill with acute nephritis. The temperature at the time was 105° F. The second was taken from a case of granular kidney in a man aged 73, who had fallen into a typhoid condition from uræmia and whose temperature had risen to 101·5° F.

The foregoing brief review of the state of the pulse in various acute diseases may throw some light upon the important law laid down by Professor Marey, as regulating the rapidity of the heart's action, namely, that increased rapidity is due to lowering, and diminished rapidity to elevation of the arterial tension. Amongst other evidence in support of this he has adduced experiments on the hearts of turtles, detached from the body, but still beating, and adapted to artificial tubes, the pressure in which can be raised or lowered at pleasure. There can be no doubt that it is proved that, other things being equal, such a relation between the arterial tension and the rate of the pulse holds good, and that variations of tension produce in this way a most important effect in the body. But Professor Marey maintains that this relation holds good almost universally, although he himself allows that one or two exceptional cases occur, such as that of peritonitis. Thus he explains the variation of the rate of pulse in different positions of the body as due solely to changes thereby produced in the fluid pressure at the level of the heart as regulated by the force of gravity. Again, he considers that the increased rapidity of the heart, which results from muscular exercise, is purely the consequence of a diminution of arterial tension caused by a relaxation of the arterioles, which allows a more ready outflow under such circumstances.

I think that a comparison of the various tracings which I have attempted to explain will tend to show that the variation

of tension is only one of the causes regulating the heart's rapidity, although it is the one most constantly in action, and that in very many cases other influences come also into play. Thus, in several other conditions besides peritonitis, pulses have been shown which are non-dicrotic and bear a high pressure, and must, therefore, indicate high arterial tension, but which, at the same time, are very rapid (*vide* Pl. I, figs. 13, 15, 31; Pl. II, figs. 22, 23, 25, 26, 29). Again, there are other pulses which are dicrotic and very compressible, but which are, nevertheless, slow enough to allow the tricrotic wave also to appear (Pl. I, figs. 2, 5, 22, 26); and it has been shown that, in the case of fever, the evidences of arterial relaxation and lowered pressure are not in uniform proportion to the increase of temperature or the rapidity of the pulse, but that, in this respect, different diseases have peculiarities of their own.

It is true that after violent exercise the pulse-curve does become more dicrotic than before, and, therefore, Professor Marey is no doubt right in supposing that the arterioles in such a case become relaxed. The increase of dicrotism, however, is not generally very great, and the tidal wave is usually still seen—an indication that the arterial tension has not fallen very low. Moreover, I have generally found in such cases that the pulse gave a higher primary upstroke under a higher pressure than it did previously to the exercise, whereas the contrary would have been the case—so far, at least, as regards the pressure—if the only change in the heart's action had been an increase of rapidity due to lowering of arterial tension. Hence the heart must have been stimulated by nervous influence to contract with greater force, and it is, therefore, probable that its increased rapidity is not solely secondary in such cases to arterial relaxation, but that it depends partly upon similar nervous influence.

The effect on the pulse of mental excitement, of alcohol, and of ether, resembles that produced by muscular exercise. In the case, however, of alcohol, and still more in that of ether, the increased vigour of the primary upstroke may be more marked. Thus the pulse which is produced by ether presents a strong contrast to the dicrotic pulse, which results from the use of nitrate of amyl, the effect of which is much greater in relaxing the vessels, but much less in stimulating the heart. The

increase of rapidity, however, may be similar in the two cases, and thus we have here another instance of corresponding increments of rapidity associated with very different states of the vascular system. Again, the pulse-curves give evidence that nervous influence may affect, not only the rapidity of the heart, but its mode of contraction; for this appears to be the only means of explaining why the curves of Bright's disease in young persons should differ from those of other high tension pulses (compare Pl. I, figs. 32, 34, 35, 37, with Pl. II, figs. 19, 20, 21); or, again, of accounting for the unusual forms of dicrotic pulse shown in Pl. I, fig. 20, and Pl. II, fig. 3. The conclusion, therefore, is that while the law stated by Professor Marey holds good so long as other conditions remain equal, yet its author has attributed to it a somewhat too universal scope, in seeking to explain by its means almost every variation in the rate of pulse.

EXPLANATION OF THE PLATES

Illustrating Dr. Galabin's Paper on the State of the Circulation in Acute Diseases.

The tracings have been copied by photo-lithography. They have been reduced in size from the originals in the proportion of three to two, as the sphygmograph with which they were taken is one whose clockwork movement is rapid, and which gives a rather greater amplitude than usual to the tracing.

PLATE I.

Fig. 1. Pulse of a healthy person, which shows the secondary waves with unusual distinctness. Pressure 3 oz.; pulse 60. The amplitude is somewhat greater than usual; the tidal wave rather more marked. The dicrotic wave is very distinct. After it is seen a slight convexity in the curve, which is probably the tricrotic wave. The condition indicated is that of somewhat relaxed vessels and a strongly acting heart. It is a kind of pulse common in those accustomed to muscular exercise.

Fig. 2. Pressure 2 oz.; temp. 100·5°; pulse 114. From a woman æt. 21, on whom the operation of excision of the knee-joint had been performed six days before on account of chronic disease. There is slight hyperdicrotism; the points of the curve are rounded, and the tricrotic wave is seen following the dicrotic.

Fig. 3. Pressure 4½ oz.; temp. 104·5°; pulse 115. From a man æt. 22, at the commencement of suppuration of the knee-joint from a penetrating wound received two days before.

Fig. 4. Pressure 4 oz.; pulse 58. From a man æt. 70, whose arteries were very rigid. The tidal wave is here greatly developed. A similar form of curve is often found in chronic Bright's disease, but in that case the pressure employed may be much greater. In both cases a prolonged contraction of the heart, in consequence of increased resistance, is indicated.

Fig. 5. Pressure 1 oz.; temp. 102·6°; pulse 98. From the same person whose pulse is shown in fig. 3, after an interval of six days. The pressure has now become very low, the amplitude small, the points of the curve rounded, and the tricrotic wave is distinctly seen.

Fig. 6. Pressure 2 oz.; temp. 104·6°; pulse 120. From a man æt. 21, on the first day of inflammatory fever, from suppuration of the bursa patellæ. The lower pressure and the more rounded points of the curve indicate a much less sthenic form of fever in this case than in that shown in fig. 3.

Fig. 7. Pressure 4 oz.; temp. 104·8°; pulse 144. From a man æt. 30, who the day before had received a penetrating wound of the chest from a crow-bar.

Fig. 8. Pressure 4 oz. Pulse of the same man fifteen minutes later, taken immediately after the abstraction of 10 oz. of blood.

Fig. 9. Pressure 3 oz.; temp. 104·7°; pulse 132. From a man æt. 44, in whose case a ligature had been placed upon the common carotid on account of hæmorrhage following an operation on the tongue.

Fig. 10. Pressure 4 oz. From the same patient as fig. 7 and fig. 8. The present tracing was taken fifteen minutes after that in fig. 8. Some reaction had then taken place; the pulse had increased in amplitude, and again become more dicrotic.

Fig. 11. Pressure 4 oz.; temp. 104·5°; pulse 102. From a boy æt. 19, on the second day of erysipelas of the face.

Fig. 12. Pressure 4 oz.; temp. 105·5°; pulse 115. From a woman æt. 23, on the third day of erysipelas of the face. Full dicrotism has barely been reached in this case. The small wave which precedes the dicrotic corresponds to the tidal wave, but occurring in this position it does not indicate the arterial tide, and may, therefore, be better called the pseudo-tidal wave. Its presence denotes a very strong and sudden action of the heart, combined with a minimum arterial tension not so low as in ordinary dicrotic pulses.

Fig. 13. Pressure 4½ oz.; temp. 103·0°; pulse 125. From a man æt. 56, and having rigid arteries, on the fourth day of erysipelas of the face.

Fig. 14. Pressure 2 oz.; temp. 99·8°; pulse 96. From the same patient, after an interval of twelve hours, during which time he had taken twenty-five minims of tincture of aconite. He was sweating profusely when this tracing was taken.

Fig. 15. Pressure 4½ oz.; temp. 101·5°; pulse 114. From a woman æt. 29, on the second day of erysipelas, following an operation upon the vulva. She was in great pain when the tracing was taken.

Fig. 16. Pressure 3½ oz.; temp. 101·6°; pulse 90. From a case of acute rheumatism in a woman æt. 25.

Fig. 17. Pressure 2 oz.; temp. 100·5°; pulse 104°. From a case of acute rheumatism, combined with mitral regurgitation, in a girl, æt. 16.

Fig. 18. Pressure 3½ oz.; temp. 102·7°; pulse 125°. From a case of peritonitis in a woman, æt. 28, who had undergone the operation of ovariotomy three weeks before. The patient died a few days after the tracing was taken.

Fig. 19. Pressure ½ oz.; temp. 104°; pulse 156. From a woman, æt. 31, who had undergone the operation of ovariotomy five days before. The tracing was taken a few hours before death. Suppuration had occurred about the wound, and the patient was much exhausted. Evidence of peritonitis was found post mortem.

Fig. 20. Pressure 3 oz.; temp. 106·2°; pulse 104. From a case of cerebral rheumatism in a woman, æt. 40. She became comatose about eight hours after the temperature had begun to rise to an unusually high point, and the tracing was taken while she was in that condition. The first of the two secondary waves is the pseudo-tidal wave, which is seen also in Pl. II, fig. 3. An unusually short jerky action of the heart is indicated by its appearance.

Fig. 21. Pressure 2½ oz.; temp. 104°; pulse 120. From a case of tubercular peritonitis in a woman, æt. 35.

Fig. 22. Pressure 1 oz.; temp. 99·6°; pulse 120. From a woman, æt. 26, who had undergone the operation of ovariotomy twenty-five days before. Post mortem, it was found that there was scarcely any peritonitis, but a rapid diffusion of malignant growth.

Fig. 23. Pressure 3 oz.; temp. 101·5°; pulse 110. From a case of pericarditis, with acute rheumatism, in a girl, æt. 13.

Fig. 24. Pressure ½ oz.; temp. 103·2°; pulse 360; resp. 65. From a case of pericarditis set up by pyæmic abscess of the heart in a girl, æt. 12. The larger elevations seen are due to the respiratory curve; the small notches are the pulsations.

Fig. 25. Pressure 2 oz.; temp. 102°; pulse 125. From a case of acute rheumatism and severe pericarditis, with effusion, in a girl, æt. 14. A fortnight later a systolic bruit at the apex became audible.

Fig. 26. Pressure 1 oz.; temp. 98·8°; pulse 120. From a case of close constriction of the mitral valve in a boy, æt. 20. The dicrotic wave is nearly as high as the primary, and the tricrotic wave is seen, although almost lost upon the succeeding upstroke.

Fig. 27. Pressure 1½ oz.; temp. 99·1°; pulse 88. From a case of bronchitis in a man, æt. 42.

Fig. 28. Pressure 1 oz.; temp. 98·4°; pulse 120. From a man, æt. 35, about two hours after hæmorrhage from the lungs to the extent of 51 oz.

Fig. 29. Pressure 1 oz.; temp. 99·6°; pulse 165. From a case of free aortic regurgitation, combined with mitral disease, in a man, æt. 22. His pulse had generally very little dicrotism, but for a short time, while the heart's action was very feeble and rapid, it became highly dicrotic.

Fig. 30. Pressure 1 oz.; temp. 98·6°; pulse 140. From a case of mitral regurgitation in a woman, æt. 20.

Fig. 31. Pressure 5 oz.; temp. 101°; pulse 136. From a case of bronchitis with albuminuria in a man, æt. 60. Post mortem, the kidneys were found pretty

healthy. The diminution of the tidal wave and descent of the base line, seen in the first pulsation, occurred with each inspiration.

Fig. 32. Pressure 4½ oz.; pulse 60. From a case of tubal nephritis of three months' standing in a boy, æt. 19.

Fig. 33. Pressure 7 oz.; pulse 94. From a case of chronic Bright's disease, in a man, æt. 50.

Fig. 34. Pressure 5 oz.; pulse 54. From a case of acute nephritis, of three weeks' duration, in a woman, æt. 25.

Fig. 35. Pressure 4 oz.; pulse 76. From a case of acute nephritis, of four weeks' duration, in a woman, æt. 23.

Fig. 36. Pressure 4 oz.; temp. 105°; pulse 110. From a girl, æt. 18, who was attacked by pleurisy and fatal purulent pericarditis, after an illness of nearly four weeks with acute nephritis.

Fig. 37. Pressure 4½ oz.; pulse 75. From a case of acute nephritis of about three weeks' duration in a man, æt. 21. In all the pulses of acute nephritis two slight waves are seen following the dicrotic wave. The first of these is formed like the tidal wave, and may be called the dicrotic tidal wave. The second of the two is either the tricrotic wave or a repetition of the last.

PLATE II.

Fig. 1. Pressure 3½ oz.; pulse 72. The usual pulse of a man, æt. 23, whose pulse is also shown in fig. 2.

Fig. 2. Pressure 2 oz.; temp. 98·2°; pulse 96. The pulse of the same man after a dose of four minims of nitrite of amyl given internally.

Fig. 3. Pressure 2½ oz.; temp. 102·8°; pulse 110. A very unusual form of pulse, obtained at the commencement of typhoid in a boy, æt. 18, whose urine at the time was albuminous.

Fig. 4. Pressure 1½ oz.; temp. 102·5°; pulse 102. The pulse of the same patient sixteen days later.

Fig. 5. Pressure 1 oz.; temp. 102·5°; pulse 150. From the same patient after two days more, and soon after the occurrence of profuse hæmorrhage, from which, however, he recovered.

Fig. 6. Pressure 1 oz.; temp. 103·4°; pulse 120. Pulse obtained on the thirteenth day of a very severe case of typhoid in a boy, æt. 13.

Fig. 7. Pressure 1 oz.; temp. 103·5°; pulse 120. Pulse obtained on the sixth day of typhoid in a girl, æt. 14.

Fig. 8. Pressure 1 oz.; temp. 103·2°; pulse 124. Pulse of a case of typhoid in a girl, æt. 13. The tracing has some resemblance to the feeble undulatory pulse of severe mitral disease shown in Pl. I, fig. 30.

Fig. 9. Pressure ½ oz.; temp. 105·5°; pulse 125. From the same patient as fig. 6, at the most severe stage of the disease. Dicrotism is here diminished simply from feebleness of the heart, although the pulse has become even more compressible than before.

Fig. 10. Pressure 1 oz.; temp. 101·9°; pulse 125. Unusual form of pulse, obtained from a boy, æt. 14, convalescent from typhoid. Primary and dicrotic waves are both very sharp, and both tidal and dicrotic tidal waves are also seen.

Fig. 11. Pressure 1 oz.; temp. 104·6°; pulse 170. From a fatal case of typhus in a man, æt. 26.

Fig. 12. Pressure 2 oz.; temp. 103·7°; pulse 105. Non-dicrotic form assumed by the pulse at the later stage of a fatal case of typhus in a man, æt. 53, whose pulse had previously been dicrotic. His urine was albuminous at the time.

Fig. 13. Pressure 2 oz.; temp. 98·1°; pulse 72. The pulse of a boy, æt. 14, convalescent from typhoid. The tricrotic wave is seen, as well as the dicrotic; the tidal is absent.

Fig. 14. Pressure 3 oz.; temp. 103·2°; pulse 104. From a woman, æt. 39, on the second day of a febrile attack, which lasted only three or four days. The presence of the tidal wave and the moderate degree of dicrotism show that there is no relaxation of arteries nor lowering of tension.

Fig. 15. Pressure 1½ oz.; temp. 103·5°; pulse 160. From a fatal case of pneumonia in a man, æt. 24, whose urine was at the time albuminous.

Fig. 16. Pressure 2 oz.; temp. 102·6°; pulse 144. From a case of pneumonia in a man, æt. 39.

Fig. 17. Pressure 3½ oz.; temp. 102·2°; pulse 110. From a fatal case of pneumonia in a man, æt. 54, whose urine was albuminous.

Fig. 18. Pressure 1 oz.; temp. 100·6°; pulse 96. From a case of phthisis, with pneumonia, in a man, æt. 35.

Fig. 19. Pressure 5½ oz.; pulse 48. From a case of fractured base of the skull in a man, æt. 54.

Fig. 20. Pressure 5 oz.; temp. 98·8°; pulse 58. From a case of abscess of the brain in a man, æt. 34.

Fig. 21. Pressure 4½ oz.; temp. 97·5°; pulse 52. Pulse showing the effect of external cold on a healthy man, æt. 26.

Fig. 22. Pressure 5 oz.; temp. 103·5°; pulse 105. From a case of comminuted fracture of the skull, exposing the brain, in a man, æt. 21.

Fig. 23. Pressure 4 oz.; temp. 99·2°; pulse 130. From a man, æt. 38, who in the last stage of hepatic ascites fell into profound coma, during which this tracing was taken. No lesion was found post mortem, except an extremely cirrhosed liver.

Fig. 24. Pressure 2 oz.; temp. 101·6°; pulse 120. From the same case as fig. 22, taken four days later. The patient at the time had continuous convulsive movements on one side.

Fig. 25. Pressure 5 oz.; pulse 114. From a case of acute mania in a girl, æt. 20.

Fig. 26. Pressure 3 oz.; temp. 105·7°; pulse 150. Pulse of a girl, æt. 11, taken during fatal coma, which succeeded to a series of epileptiform fits. No lesion was found post mortem.

Fig. 27. Pressure 2½ oz.; temp. 101·6°; pulse 110. From a case of uræmia from chronic Bright's disease in a man, æt. 73.

Fig. 28. Pressure 4½ oz.; pulse 60. From a case of subacute mania in a woman, æt. 25.

Fig. 29. Pressure 4½ oz.; pulse 110. From a case of subacute mania in a woman, æt. 50.

Fig. 30. Pressure 2½ oz.; temp. 103·2°; pulse 145. The pulse of a woman,

æt. 30, taken when she was in a comatose condition from tubercular meningitis, which came on in the course of phthisis.

Fig. 31. Pressure 2 oz.; temp. 105·3°; pulse 144. From a case of extensive sclerosis of the brain and spinal cord in a woman, æt. 23. The tracing was taken about two hours before death, after she had suddenly become comatose. The respiratory curve is marked, and one or two beats are much enfeebled by the effect of a gasp at the end of inspiration.

Fig. 32. Pressure 2½ oz.; pulse 120. Taken from the same person as fig. 29, on a different occasion.

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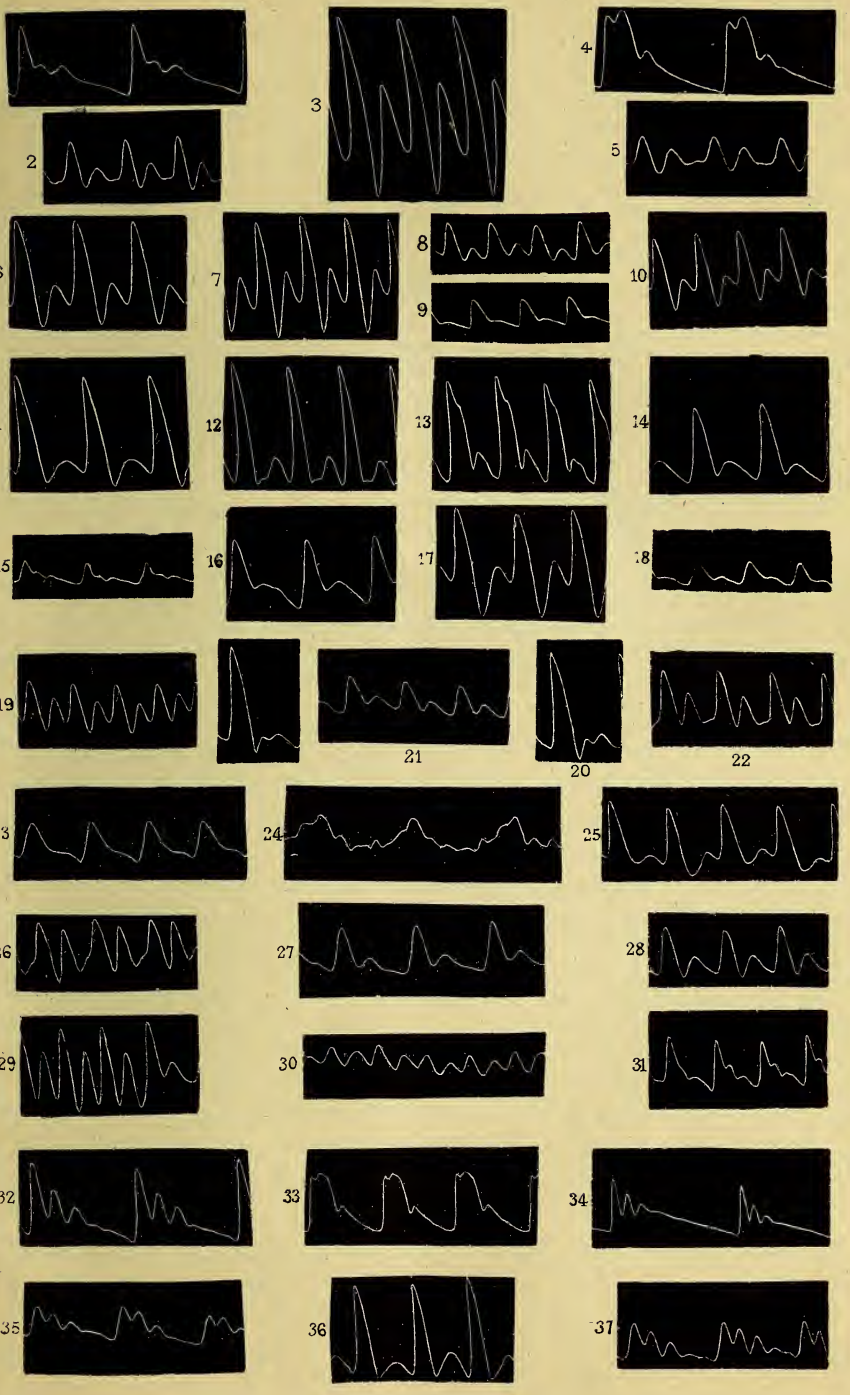

Whiteman & Bass, *Photo Litho to the Queen*, Holborn, London

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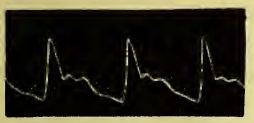
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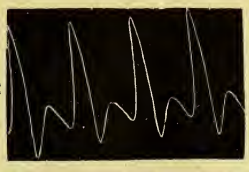
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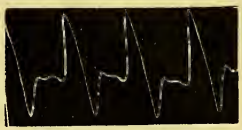
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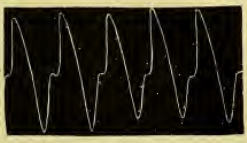
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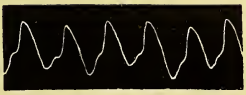
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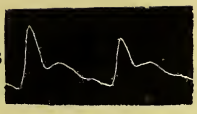
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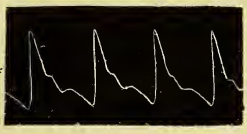
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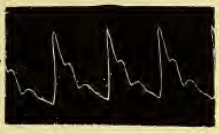
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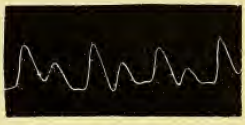
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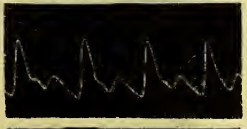
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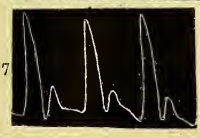
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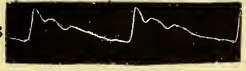
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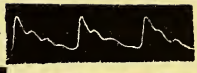
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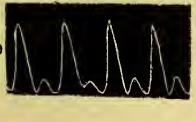
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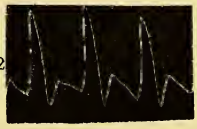
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Whitehead & Bass, *Photo Litho to the Queen*, Holborn, London.